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L1	deiss-louis.in.	0	L1

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☐ 1. Document ID: US 6057111 A

L2: Entry 1 of 2 File: USPT May 2, 2000

US-PAT-NO: 6057111
DOCUMENT-IDENTIFIER: US 6057111 A

TITLE: Gene identification method

DATE-ISSUED: May 2, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deiss; Louis Paul	Chicago	IL		
Yehiely; Fruma	Chicago	IL		
Efimova; Elena	Chicago	IL		
Vasquez-Iaslop; Nora Cecilia	Oak Park	IL		
Einat; Paz	Nes Ziona			IL

US-CL-CURRENT: 435/6; 435/29, 435/320.1

ABSTRACT:

A method for the identification of genes that are essential for the maintenance of specific cell phenotypes is disclosed. The method includes the initial step of identifying a cell type with a phenotype of interest. Gene inactivation is performed on an aliquot of cells of the cell type of interest. Positive selection is then performed to an aliquot of the cell culture to which gene inactivation has been applied. Cells which continue to maintain the phenotype following gene inactivation have not had the gene of interest inactivated whereas cells in which genes necessary for maintaining the phenotype have been inactivated have been lost. Utilizing subtraction analysis between treated and untreated aliquots the gene in the cells which has been inactivated that affects the phenotype of interest is identified. Genes that are identified by the method are also disclosed as well as antibodies directed against the gene product of the identified genes. Further a customized kit for the practice of the gene identification method is also disclosed.

7 Claims, 2 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 2

L2: Entry 1 of 2 File: USPT May 2, 2000

DOCUMENT-IDENTIFIER: US 6057111 A
TITLE: Gene identification method

INVENTOR (1):
Deiss; Louis Paul

☐ 2. Document ID: WO 9821366 A1

L2: Entry 2 of 2 File: EPAB May 22, 1998

PUB-NO: WO009821366A1
DOCUMENT-IDENTIFIER: WO 9821366 A1
TITLE: GENE IDENTIFICATION METHOD

PUBN-DATE: May 22, 1998

INVENTOR-INFORMATION:

NAME	COUNTRY
DEISS, LOUIS PAUL	US
YEHIELY, FRUMA	US
EFIMOVA, ELENA	US
VASQUEZ-IASLOP, NORA CECILIA	US
EINAT, PAZ	IL

INT-CL (IPC): C12 Q 1/68; C12 Q 1/02; C07 H 21/04
EUR-CL (EPC): C12Q001/68; C12N015/10, C12Q001/02

ABSTRACT:

CHG DATE=19990617 STATUS=O>A method for the identification of genes that are essential for the maintenance of specific cell phenotypes is disclosed. The method includes the initial step of identifying a cell type with a phenotype of interest. Gene inactivation is performed on an aliquot of cells of the cell type of interest. Positive selection is then performed to an aliquot of the cell culture to which gene inactivation has been applied. Cells which continue to maintain the phenotype following gene inactivation have not had the gene of interest inactivated whereas cells in which genes necessary for maintaining the phenotype have been inactivated have been lost. Utilizing subtraction analysis between treated and untreated aliquots the gene in the cells which has been inactivated that affects the phenotype of interest is identified. Genes that are identified by the method are also disclosed as well as antibodies directed against the gene product of the identified genes. Further a customized kit for the practice of the gene identification method is also disclosed.

L2: Entry 2 of 2 File: EPAB May 22, 1998

DOCUMENT-IDENTIFIER: WO 9821366 A1
TITLE: GENE IDENTIFICATION METHOD

Inventor Name (Derived) (1):
DEISS, LOUIS PAUL

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Term	Documents
DEISS-LOUIS$	0
DEISS-LOUIS-PAUL.DWPI,EPAB,JPAB,USPT,PGPB.	2
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L3 ANSWER 1 OF 6 MEDLINE DUPLICATE 1
AN 2003073771 MEDLINE
DN 22472493 PubMed ID: 12584558
TI Nrf2 is an inhibitor of the Fas pathway as identified by Achilles' Heel Method, a new function-based approach to gene identification in human cells.
AU Kotlo Kumar U; Yehiely Furma; Efimova Elena; Harasty Heather; Hesabi Bahar; Shchors Ksenya; Einat Paz; Rozen Ada; Berent Eva; **Deiss Louis P**
CS Department of Molecular Genetics (M/C 669), University of Illinois at Chicago, 60607, USA.
SO ONCOGENE, (2003 Feb 13) 22 (6) 797-806.
Journal code: 8711562. ISSN: 0950-9232.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20030214
Last Updated on STN: 20030304
Entered Medline: 20030303
AB Here we describe the Achilles' Heel Method (AHM), a new function-based approach for identification of inhibitors of signaling pathways, optimized for human cells. The principle of AHM is the identification of 'sensitizing' cDNAs based on their decreased abundance following **selection**. As a proof of principle, we have employed AHM for the identification of Fas/CD95/APO-1 pathway inhibitors. HeLa cells were transfected with an antisense cDNA expression library in an episomal vector followed by **selection** with a suboptimal dose of the apoptotic inducer. Antisense inactivation of Fas inhibitors rendered the cells more sensitive to apoptosis resulting in their preferential death and consequent loss of their sensitizing episomes that were identified by subtraction. We show that the resulting products were enriched for sensitizing cDNAs as seven out of eight candidates tested were confirmed as inhibitors of Fas-induced killing either by transfection or by pharmacological inhibition. Furthermore, we demonstrate by multiple approaches that one candidate, NF-E2 related factor 2 (Nrf2), is an inhibitor of Fas-induced apoptosis. Inactivation of Nrf2 by antisense or by a membrane permeable dominant-negative polypeptide sensitized cells while overexpression of Nrf2 protected cells from Fas-induced apoptosis. In addition, dicumarol, an inhibitor of the phase II detoxifying enzyme NQO1, a downstream target of Nrf2, sensitized cells. Nrf2 induces the production of Glutathione (GSH) and we demonstrated that N-acetyl L-cysteine (NAC), a precursor to GSH, protected cells from Fas-mediated killing. Taken together, AHM is a powerful approach for the identification of inhibitors of a signaling pathway with a low rate of false positives that opens new avenues for function profiling of human genes and discovery of new drug targets.

L3 ANSWER 2 OF 6 MEDLINE DUPLICATE 2
AN 96324394 MEDLINE
DN 96324394 PubMed ID: 8670891
TI Cathepsin D protease mediates programmed cell death induced by interferon-gamma, Fas/APO-1 and TNF-alpha.
AU **Deiss L P**; Galinka H; Berissi H; Cohen O; Kimchi A
CS Department of Molecular Genetics and Virology, Weizmann Institute of Science, Rehovot 76100, Israel.
SO EMBO JOURNAL, (1996 Aug 1) 15 (15) 3861-70.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199610
ED Entered STN: 19961022
Last Updated on STN: 19970203
Entered Medline: 19961010
AB A functional approach of gene cloning was applied to HeLa cells in an attempt to isolate positive mediators of programmed cell death. The approach was based on random inactivation of genes by transfections with antisense cDNA expression libraries, followed by the **selection** of cells that survived in the presence of the external apoptotic stimulus. An antisense cDNA fragment identical to human cathepsin D aspartic protease was rescued by this positive **selection**. The high cathepsin D antisense RNA levels protected the HeLa cells from interferon-gamma- and Fas/APO-1-induced death. Pepstatin A, an inhibitor of cathepsin D, suppressed cell death in these systems and interfered with the TNF-alpha-induced programmed cell death of U937 cells as well. During cell death, expression of cathepsin D was elevated and processing of the protein was affected, which resulted in high steady-state levels of an intermediate, proteolytically active, single chain form of this protease. Overexpression of cathepsin D by ectopic expression induced cell death in the absence of any external stimulus. Altogether, these results suggest that this well-known endoprotease plays an active role in cytokine-induced programmed cell death, thus adding cathepsin D to the growing list of proteases that function as positive mediators of apoptosis.

L3 ANSWER 3 OF 6 MEDLINE DUPLICATE 3
AN 96070931 MEDLINE
DN 96070931 PubMed ID: 7499268
TI Isolation of DAP3, a novel mediator of interferon-gamma-induced cell death.
AU Kissil J L; **Deiss L P**; Bayewitch M; Raveh T; Khaspekov G; Kimchi A
CS Department of Molecular Genetics and Virology, Weizmann Institute of Science, Rehovot, Israel.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Nov 17) 270 (46) 27932-6.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X83544
EM 199601
ED Entered STN: 19960217
Last Updated on STN: 19970203
Entered Medline: 19960117
AB Interaction of certain cytokines with their corresponding cell-surface receptors induces programmed cell death. Interferon-gamma induces in HeLa cells a type of cell death with features characteristic of programmed cell death. Here, we report the isolation of a novel gene, DAP3 (death-associated protein-3), involved in mediating interferon-gamma-induced cell death. The rescue of this gene was performed by a functional **selection** approach of gene cloning that is based on transfection with an antisense cDNA expression library. The antisense RNA-mediated inactivation of the DAP3 gene protected the cells from interferon-gamma-induced cell death. This property endowed the cells expressing it with a growth advantage in an environment restrictive due to the continuous presence of interferon-gamma and thus provided the basis of its **selection**. The gene is transcribed into a single 1.7-kilobase mRNA, which is ubiquitously expressed in different tissues and codes for a 46-kDa protein carrying a potential P-loop motif. Ectopic expression of DAP3 in HeLa cells was not compatible with cell growth, resulting in a 16-fold reduction in the number of drug-resistant stable

clones. The data presented suggest that DAP3 is a positive mediator of cell death induced by interferon-gamma.

L3 ANSWER 4 OF 6 MEDLINE DUPLICATE 4
AN 95129831 MEDLINE
DN 95129831 PubMed ID: 7828849
TI Identification of a novel serine/threonine kinase and a novel 15-kD protein as potential mediators of the gamma interferon-induced cell death.
AU **Deiss L P**; Feinstein E; Berissi H; Cohen O; Kimchi A
CS Department of Molecular Genetics and Virology, Weizmann Institute of Science, Rehovot, Israel.
SO GENES AND DEVELOPMENT, (1995 Jan 1) 9 (1) 15-30.
Journal code: 8711660. ISSN: 0890-9369.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X76104; GENBANK-X76105
EM 199502
ED Entered STN: 19950307
Last Updated on STN: 19980206
Entered Medline: 19950221
AB Programmed cell death is often triggered by the interaction of some cytokines with their cell surface receptors. Here, we report that gamma interferon (IFN-gamma) induced in HeLa cells a type of cell death that had cytological characteristics of programmed cell death. In this system we have identified two novel genes whose expression was indispensable for the execution of this type of cell death. The rescue was based on positive growth **selection** of cells after transfection with antisense cDNA expression libraries. The antisense RNA-mediated inactivation of the two novel genes protected the cells from the IFN-gamma-induced cell death but not from the cytostatic effects of the cytokine or from a necrotic type of cell death. One of those genes (DAP-1) is expressed as a single 2.4-kb mRNA that codes for a basic, proline-rich, 15-kD protein. The second is transcribed into a single 6.3-kb mRNA and codes for a unique 160-kD calmodulin-dependent serine/threonine kinase (DAP kinase) that carries eight ankyrin repeats. The expression levels of the two DAP proteins were selectively reduced by the corresponding antisense RNAs. Altogether, it is suggested that these two novel genes are candidates for positive mediators of programmed cell death that is induced by IFN-gamma.

L3 ANSWER 5 OF 6 MEDLINE DUPLICATE 5
AN 91188270 MEDLINE
DN 91188270 PubMed ID: 1901424
TI A genetic tool used to identify thioredoxin as a mediator of a growth inhibitory signal.
AU **Deiss L P**; Kimchi A
CS Department of Molecular Genetics and Virology, Weizmann Institute of Science, Rehovot, Israel.
SO SCIENCE, (1991 Apr 5) 252 (5002) 117-20.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199105
ED Entered STN: 19910526
Last Updated on STN: 19970203
Entered Medline: 19910507
AB Loss of sensitivity to growth inhibitory polypeptides is likely to be one of the events that participates in the formation of some tumors and might be caused by inactivation or loss of the genetic elements that transduce these extracellular signals. The isolation of such a gene was achieved by randomly inactivating genes by an anti-sense complementary DNA expression

library followed by direct **selection** for growth in the presence of an inhibitory polypeptide. Thus, a gene whose inactivation conveyed growth resistance to interferon-gamma (IFN-gamma) was isolated. Sequence analysis showed complete identity with human thioredoxin, a dithiol reducing agent, implicated here in the IFN-gamma-mediated growth arrest of HeLa cells.

L3 ANSWER 6 OF 6 MEDLINE DUPLICATE 6
AN 86281869 MEDLINE
DN 86281869 PubMed ID: 3016323
TI Functional domains within the a sequence involved in the cleavage-packaging of herpes simplex virus DNA.
AU **Deiss L P**; Chou J; Frenkel N
NC AI-15488 (NIAID)
CA-09241 (NCI)
CA-19264 (NCI)
+
SO JOURNAL OF VIROLOGY, (1986 Sep) 59 (3) 605-18.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M13884; GENBANK-M13885
EM 198609
ED Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860917
AB Newly replicated herpes simplex virus (HSV) DNA consists of head-to-tail concatemers which are cleaved to generate unit-length genomes bounded by the terminally reiterated a sequence. Constructed defective HSV vectors (amplicons) containing a viral DNA replication origin and the a sequence are similarly replicated into large concatemers which are cleaved at a sequences punctuating the junctions between adjacent repeat units, concurrent with the packaging of viral DNA into nucleocapsids. In the present study we tested the ability of seed amplicons containing specific deletions in the a sequence to become cleaved and packaged and hence be propagated in virus stocks. These studies revealed that two separate signals, located within the Ub and Uc elements of the a sequence, were essential for amplicon propagation. No derivative defective genomes were recovered from seed constructs which lacked the Uc signal. In contrast, propagation of seed constructs lacking the Ub signal resulted in the **selection** of defective genomes with novel junctions, containing specific insertions of a sequences derived from the helper virus DNA. Comparison of published sequences of concatemeric junctions of several herpesviruses supported a uniform mechanism for the cleavage-packaging process, involving the measurement from two highly conserved blocks of sequences (pac-1 and pac-2) which were homologous to the required Uc and Ub sequences. These results form the basis for general models for the mechanism of cleavage-packaging of herpesvirus DNA.

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